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New mechanism of acute kidney injury pathological role of arteriovenous anastomoses

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The problem of acute kidney injury (AKI) is very complicated. The main causes of AKI are physical interactions. It leads to blood stagnation. The main factor contributing to stagnation of blood, are functioning arteriovenous anastomoses (AVA). At the first stage, with an increase in blood pressure, arterial deformities can occur: increased arterial crimp in kidney, collateral growth, and increase in arterial stiffness, and growth of plaques. In addition, AVA's can open when a significant increase in blood pressure. Wherein BP falls, but the venous pressure increases. The balance of arterial and venous blood volume is disturbed. If there are valves absent or there are valves damaged by pressure, in the venous pressure can be transferred from one organ to another. For example, if there are AVA's between the superior mesenteric artery and portal vein, it can lead increase venous pressure in the renal veins. An increase in venous pressure can lead to a decrease in the pressure gradient between arterioles and venules in the kidneys. This leads to stagnation, retardation and perfusion stasis in the kidneys, and a decrease in the release of urine. I believe that official medicine needs to focus its efforts on a thorough study of the AVA's work under various conditions for their growth and functioning. It is necessary to develop a new system of measures to prevent AKI, venous stasis, CVD..

Biography

Vladimir Ermoshkin has graduated in Physics Department of Lomonosov Moscow State University in 1978. He had worked in RosNOU, physicist. Starting in 2011, He have published about 20 articles on cardiology in prominent magazines (Russian and English), and some time He had speech at international medical conferences.

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